

	Type	L #	Hits	Search Text	Dbs	Time Stamp	Comments	Error Definition	Error
1	BRS	L1	55	pr-39	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/3 0 17:51			0
2	BRS	L3	3	pr-39 same oligopeptide	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/3 0 17:52			0
3	BRS	L4	465	proteasome	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/3 0 17:53			0
4	BRS	L5	210	4 same (inhibition or inhibitor)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/3 0 17:53			0
5	BRS	L6	2	1 same 5	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/3 0 17:53			0

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA'  
ENTERED AT

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L1 364 S PR-39

L2 21657 S PROTEASOME

L3 9946 S L2 (P) INHIBIT?

L5 9 S L1 (P) L3

L6 6 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)

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=> s pr-39  
L1 364 PR-39

=> s proteasome  
L2 21657 PROTEASOME

=> s l2 (p) inhibit?  
L3 9946 L2 (P) INHIBIT?

=> s l1 (p) l1  
L4 364 L1 (P) L1

=> s l1 9p) l3  
MISSING OPERATOR L1 9P  
The search profile that was entered contains terms or  
nested terms that are not separated by a logical operator.

=> s l1 (p) l3  
L5 9 L1 (P) L3

=> duplicate remove l5  
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'  
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n  
PROCESSING COMPLETED FOR L5  
L6 6 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)

=> d l6 1-6 ibib abs

L6 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2001:489246 CAPLUS  
DOCUMENT NUMBER: 135:87168  
TITLE: Method for PR-39 peptide-mediated selective inhibition  
of I.kappa.B.alpha. degradation  
INVENTOR(S): Simons, Michael; Gao, Youhe  
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA  
SOURCE: PCT Int. Appl., 68 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2001047540	A1	20010705	WO 2000-US35293	20001227

W: AU, CA, JP  
RW: AT, BE, CH, CY, DE, ES, FI, FR, GB, GR, IE, IT, L MC, NL,  
PT, SE, TR

PRIORITY APPLN. INFO.: US 1999-474967 A 19991229

AB The invention provides both a method and means for regulating I.kappa.B.alpha. degrdn., NF.kappa.B activity, and NF.kappa.B-dependent gene expression within living cells, tissues, and organs in-situ. The selective regulation is performed using native PR-39 peptide or one of its shorter-length homologs, for interaction with such I.kappa.B.alpha. and proteasomes as are present in the cytoplasm of viable cells. The result of PR-39 peptide interaction with I.kappa.B.alpha. is a selective alteration in the intracellular proteolytic activity of proteasomes, which in turn, causes a redn. of I.kappa.B.alpha., a decrease of NF.kappa.B activity, and a down-regulation of NF.kappa.B-dependent gene expression.

L6 ANSWER 2 OF 6 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 2001663431 MEDLINE  
DOCUMENT NUMBER: 21565666 PubMed ID: 11709430  
TITLE: \*\*\*PR\*\*\* - \*\*\*39\*\*\* and PR-11 peptides  
\*\*\*inhibit\*\*\* ischemia-reperfusion injury by blocking  
\*\*\*proteasome\*\*\* -mediated I kappa B alpha degradation.  
AUTHOR: Bao J; Sato K; Li M; Gao Y; Abid R; Aird W; Simons M; Post M J  
CORPORATE SOURCE: Angiogenesis Research Center, Dartmouth Medical School, Hanover, New Hampshire 03756, USA.  
CONTRACT NUMBER: HL-53793 (NHLBI)  
HL-636-09 (NHLBI)  
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY. HEART AND CIRCULATORY PHYSIOLOGY, (2001 Dec) 281 (6) H2612-8.  
Journal code: 100901228. ISSN: 0363-6135.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200201  
ENTRY DATE: Entered STN: 20011119  
Last Updated on STN: 20020125  
Entered Medline: 20020107

AB \*\*\*PR\*\*\* - \*\*\*39\*\*\* \*\*\*inhibits\*\*\* \*\*\*proteasome\*\*\* -mediated I kappa B alpha degradation and might protect against ischemia-reperfusion injury. We studied \*\*\*PR\*\*\* - \*\*\*39\*\*\*, its truncated form PR-11, and a mutant PR-11AAA, which lacks the ability to prevent I kappa B alpha degradation, in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg \*\*\*PR\*\*\* - \*\*\*39\*\*\* or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, respectively, which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility (+/-dP/dt) compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion molecule-1 and vascular cell adhesion molecule 1 were reduced. Thus \*\*\*PR\*\*\* - \*\*\*39\*\*\* and PR-11 effectively \*\*\*inhibit\*\*\* myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by \*\*\*inhibition\*\*\* of I kappa B alpha degradation and subsequent \*\*\*inhibition\*\*\* of nuclear factor-kappa B-dependent adhesion molecules. The active sequence is located in the first 11 amino acids, suggesting a potential for oligopeptide therapy as an adjunct to revascularization.

L6 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2001:936421 CAPLUS  
DOCUMENT NUMBER: 136:178301  
TITLE: \*\*\*PR\*\*\* - \*\*\*39\*\*\* and PR-11 peptides  
\*\*\*inhibit\*\*\* ischemia-reperfusion injury by blocking \*\*\*proteasome\*\*\* -mediated I.kappa.B.alpha. degradation  
AUTHOR(S): Bao, Jialin; Sato, Kaori; Li, Min; Gao, Youhe; Abid, Ruhul; Aird, William; Simons, Michael; Post, Mark J.  
CORPORATE SOURCE: Angiogenesis Research Center, Beth Israel Deaconess Medical Center, Dartmouth Medical School, Hanover, NH, 03756, USA  
SOURCE: American Journal of Physiology (2001), 281(6, Pt. 2),

PUBLISHER: American Physiological Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB \*\*\*PR\*\*\* - \*\*\*39\*\*\* \*\*\*inhibits\*\*\* \*\*\*proteasome\*\*\* -mediated

I.kappa.B.alpha. degrdn. and might protect against ischemia-reperfusion injury. The authors studied \*\*\*PR\*\*\* - \*\*\*39\*\*\*, its truncated form PR-11, and a mutant PR-11AAA, which lacks the ability to prevent I.kappa.B.alpha. degrdn., in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg \*\*\*PR\*\*\* - \*\*\*39\*\*\* or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, resp., which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion mol.-1 and vascular cell adhesion mol. 1 were reduced. Thus, \*\*\*PR\*\*\* - \*\*\*39\*\*\* and PR-11 effectively \*\*\*inhibit\*\*\* myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by \*\*\*inhibition\*\*\* of I.kappa.B.alpha. degrdn. and subsequent \*\*\*inhibition\*\*\* of nuclear factor-kappa.B-dependent adhesion mols. The active sequence is located in the first 11 amino acids, suggesting a potential for oligopeptide therapy as an adjunct to revascularization.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 2001:935656 SCISEARCH

THE GENUINE ARTICLE: 487UW

TITLE: \*\*\*PR\*\*\* - \*\*\*39\*\*\* and PR-11 peptides protect against ischemia-reperfusion injury by \*\*\*inhibition\*\*\* of \*\*\*proteasome\*\*\* mediated I kappa B alpha degradation

AUTHOR: Bao J L (Reprint); Gao Y H; Li M; Abid M R; Aird W; Simons M; Post M J

CORPORATE SOURCE: Harvard Univ, Beth Israel Deaconess Med Ctr, Sch Med, Boston, MA 02215 USA

COUNTRY OF AUTHOR: USA

SOURCE: CIRCULATION, (23 OCT 2001) Vol. 104, No. 17, Supp. [S], pp. 52-52. MA 251.

Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.

ISSN: 0009-7322.

DOCUMENT TYPE: Conference; Journal

LANGUAGE: English

REFERENCE COUNT: 0

L6 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:178321 CAPLUS

DOCUMENT NUMBER: 133:205925

TITLE: PR39, a peptide regulator of angiogenesis. [Erratum to document cited in CA132:149677]

AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; Gao, Youhe; Li, Min; Metals, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; Simons, Michael

CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA

SOURCE: Nature Medicine (New York) (2000), 6(3), 356

CODEN: NAMEFI; ISSN: 1078-8956

PUBLISHER: Nature America

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The correct versions are given for Figs. 2a, c, and d on page 51; Fig. 3c on page 52; and Fig. 5b on page 53.

L6 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:46162 CAPLUS

DOCUMENT NUMBER: 132:149677  
 TITLE: PR39, a peptide regulator of angiogenesis  
 AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; Gao, Youhe; Li, Min; Metais, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; Simons, Michael  
 CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery both at Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA  
 SOURCE: Nat. Med. (N. Y.) (2000), 6(1), 49-55  
 CODEN: NAMEFI; ISSN: 1078-8956  
 PUBLISHER: Nature America  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB Although tissue injury and inflammation are considered essential for the induction of angiogenesis, the mol. controls of this cascade are mostly unknown. Here we show that a macrophage-derived peptide, PR39, inhibited the ubiquitin-proteasome-dependent degrdn. of hypoxia-inducible factor-1.alpha. protein, resulting in accelerated formation of vascular structures in vitro and increased myocardial vasculature in mice. For the latter, coronary flow studies demonstrated that PR39-induced angiogenesis resulted in the prodn. of functional blood vessels. These findings show that PR39 and related compds. can be used as potent inducers of angiogenesis, and that selective inhibition of hypoxia-inducible factor-1.alpha. degrdn. may underlie the mechanism of inflammation-induced angiogenesis.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L1 364 S PR-39  
 L2 21657 S PROTEASOME  
 L3 9946 S L2 (P) INHIBIT?  
 L4 364 S L1 (P) L1  
 L5 9 S L1 (P) L3  
 L6 6 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)

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